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Conditioned Heart Rate as a Function of Anxiety and CS-UCS Interval

by

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Technical Report 1

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Foreword

This investigation was carried out as part of a project concerned with the influence of motivation on performance in learning under Contract N9 onr-93802, Project NR 154-107 between the State University of Iowa and the Office of Naval Research.

Acknowledgement is made to Dr. Elaine Taylor for her aid in running subjects, and to Dr. R. Beecroft, Mr. R. Czeh, and Miss Rhoda Ketchel for their aid in analyzing the data.

A list of the reports made thus far in connection with this project is given on page 21.

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A number of studies (2, 3, 4, 5, 6, 14, 15) have indicated that changes in heart rate can be acquired and extinguished, in the manner of other conditioned responses. The present study represents a further investigation of the conditioned human heart rate, in relation to two variables that have been shown to influence level of performance of other conditioned responses: manifest anxiety (8, 9, 10); and length of the CS-UCS interval (1, 7, 12, 13).

Method

Apparatus. Ss were run in a 6x10 ft. semi-soundproof room having a constant illumination of 0.15 apparent ft. candles. They were seated in an adjustable dental chair facing a circular milk-glass disk 2 3/4 in. in diameter, located slightly above eye level, 92 in. in front of S. The CS, whose duration was respectively either 500 or 5000 msec. for different groups, consisted of an increase in the brightness of this disk from 0.085 to 14.56 apparent ft. candles. The UCS was an electric shock provided by the discharge of a four-microfarad condenser, administered during the last 50 msec. of the CS to the tips of the index and middle fingers of S's left hand by means of small silver electrodes.

Heart beat was recorded electrically by means of curved EKG electrodes fastened to the palmar side of each of S's wrists, and connected

to an amplifier system which transmitted the strengthened signal to a brush BL 902 pen recorder and polygraph. E was located in a second room containing the recording apparatus and stimulus controls.

Subjects. Ss were 133 students from an introductory course in psychology. Of these, 65 constituted a high anxiety (HA) group, having obtained scores within the upper 20% of scores of a standardization population on the Taylor Anxiety Scale (11). The remaining 68 Ss were designated the low anxiety (LA) group, having obtained scores within the lower 20% of the Taylor Scale. The CS-UCS interval of 500 msec. was used for 34 HA and for 35 LA Ss. The remaining Ss, 31 HA and 33 LA, were conditioned at the 5000 msec. CS-UCS interval.

Procedure. Before the beginning of the experiment, the level of shock to be used for each S was determined by giving a series of shocks, starting at 20 v. and increasing in 10 v. steps, until it was reported that the shock was "definitely unpleasant but not painful." The shock was maintained at that level during the entire experiment.[1] Level of shock was not increased beyond 110 v. for any S. The range of shock utilized for all Ss was from 20 v. to 110 v. Mean voltages for the four groups during the experiment were: 500-HA, 52.9; 500-LA, 52.6; 5000-HA, 49.1; 5000-LA, 54.1.

Following the voltage adjustment, each S was given ten preliminary

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[1] Preliminary investigation indicated that the effect upon heart rate of increasing shock intensity as the experiment progressed did not differ from that of maintaining shock at a constant level throughout.

pre-conditioning trials in which either the CS (light) or UCS (shock) was presented alone in a predetermined order (L S S L L S L S S L). A weak buzzer was sounded prior to the presentation of any stimulus, as a ready signal, indicating that S was to fixate the disk. For the 500 msec. CS-UCS group, the ready signal was given irregularly 6.5, 7.5, or 8.5 sec. before the onset of the CS. The intervals for the 5000 msec. CS-UCS group were given irregularly 2, 3, or 4 sec. before the CS. This procedure, which was followed throughout the remainder of the experiment, insured a comparable mean interval between the ready signal and UCS for both the 500 and 5000 msec. groups.

Forty conditioning trials were then given. Since the CS-UCS interval for the 500 msec. condition was too short to permit measurement of a conditioned response prior to the onset of the UCS, the UCS was omitted on eight of these trials. The eight test trials were administered to each S according to one of three schedules, each of which involved the presentation of succeeding test trials in an irregular fashion, following blocks of 4, 5, or 6 actual conditioning trials.

The inter-trial interval varied from 25 to 35 sec., with an average of 30 sec. After every sixth trial the inter-trial interval was lengthened to 55-65 sec., with an average of 60 sec., to permit S to change his position if he wished.

Following the conditioning series, ten extinction trials were given, in which only the CS was presented.

Response measures. S's heartbeat was recorded for several seconds

before and following each presentation of the CS, long enough to obtain a record of at least five pulses (four cycles) both before and after the onset of the CS. Since a constant speed polygraph was used, the distance (in mm.) between heart beats could be converted into beats per min. The effect of the CS on a given trial was measured by subtracting the heart rate, in beats per min., during the four cycles immediately preceding the onset of the CS (Pre-CS) from the rate during the four cycles immediately following the CS onset (Post-CS).[2] Thus, a positive Post-CS minus Pre-CS score would indicate an accelerating effect of CS on heart rate and a negative score a decelerating effect.

On an occasional trial, the polygraph records were not readable, usually because of the recording of action potentials resulting from S's movements. In processing the data, when a measure could not be secured, an estimated value was obtained by averaging the immediately preceding and immediately succeeding measures.

Results and Discussion

Initial response to UCS. In order to evaluate the effect of the UCS, the Pre-UCS heart rate was subtracted from the Post-UCS heart rate.

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[2] The term "Post-CS," it should be noted, has somewhat different meanings when applied to the 500 msec. and to the 5000 msec. conditions. In the 5000 msec. CS groups, the CS was usually on during the entire four cycle period following CS onset, whereas in the 500 msec. CS groups, it was on for only a fraction of the period, or not at all, during the four cycles following CS onset.

Since this effect was confounded with that of the CS during the conditioning series, it was analyzed for only the second trial of the preliminary series, the first occasion on which shock alone was presented. In this single instance heart rate was computed for only two cycles before and after the UCS, in order to determine the effect of the UCS at a point relatively close to the time of its offset, in accordance with a suggestion by Zeaman and Wegner (15). The resulting scores for the four groups of Ss are presented in Table 1. They clearly indicate that shock produced a marked acceleration in heart rate in all groups, a result consistent with previous findings (15). Level of anxiety was not related to the magnitude of acceleration. However, the combined 500-msec. groups (M = 9.66, SD = 8.33) showed reliably greater acceleration than the combined 5000 msec. groups (M = 4.35, SD = 6.89), the difference yielding a t-value of 3.99, $p < .001$.

Initial Response to CS. The effect of CS alone is shown in Table 2 which gives the Post-CS minus Pre-CS scores for the four groups of Ss on the first trial of the preliminary series, when only the light was presented. These data indicate that the initial effect of the 5000 msec. CS was a retardation of heart rate, whereas the 500 msec. CS had no reliable effect. As in the case of the response to the UCS, no differences were found between anxiety levels. When anxiety levels within the two CS-UCS conditions were combined, the 5000 msec. group (M = -2.92, SD = 5.28) showed significantly greater deceleration in heart rate than did the 500 msec. group (M = -0.23, SD = 5.28), with t = 2.92, $p < .01$.

The explanation of the difference between the two CS conditions can

Table 1

Effect of UCS on Heart Rate (Post-UCS minus Pre-UCS rate)

On First Preliminary Trial Involving UCS Only

Group	N	M	SD	t	p
500-HA	34	8.74	8.98	5.60	$<.001$
500-LA	35	10.55	7.55	8.11	$<.001$
5000-HA	31	5.51	6.86	4.40	$<.001$
5000-LA	33	3.26	6.75	2.71	$<.02$

Table 2

Effect of CS on Heart Rate (Post-CS minus Pre-CS rate)

On First Preliminary Trial Involving CS Only

Group	N	M	SD	t	p
500-HA	34	-0.22	5.13	0.24	$<.90$
500-LA	35	-0.24	5.43	0.25	$<.90$
5000-HA	31	-3.68	5.13	3.91	$<.001$
5000-LA	33	-2.21	5.31	2.35	$<.05$

only be surmised. Since there is evidence (14, 15) that auditory stimulation may depress heart rate, it is possible that the initial retardation in rate shown by the 5000 msec. groups is attributable to the occurrence of the buzzer (ready signal) 2-4 sec. prior to the onset of the light. In the 500 msec. groups, the ready signal preceded the CS by a longer interval, 6.5-8.5 sec. The differential effect of the UCS upon the responses of the 500 vs. the 5000 msec. group must have been due, in some way, to the differential effect of the first CS trial on these groups, since the conditions under which the UCS was presented were identical.

Changes in Response to CS. Performance curves relating effect of CS to practice during the preliminary series (CS only), during conditioning, and during extinction are presented in Fig. 1. These curves were constructed by averaging the medians of each trial within overlapping blocks of three trials. Thus, the mean of the medians was calculated for trials 1, 4, 5, for trials 4, 5, 7, and for trials 5, 7, 10 of the preliminary series, during which CS alone was presented. It may be seen from these data that, in the 500 msec. groups, the initial acceleration due to presentation of the CS was further increased, and that in the 5000 msec. groups, the initial deceleration was further increased, during the preliminary trials.

Table 3 presents the mean of the means for trials 1 and 4 and for trials 5, 7, and 10 of the preliminary series. It indicates also the significance of the deviation of each mean from zero (i.e., no effect of CS on heart rate), as well as the significance of the difference between

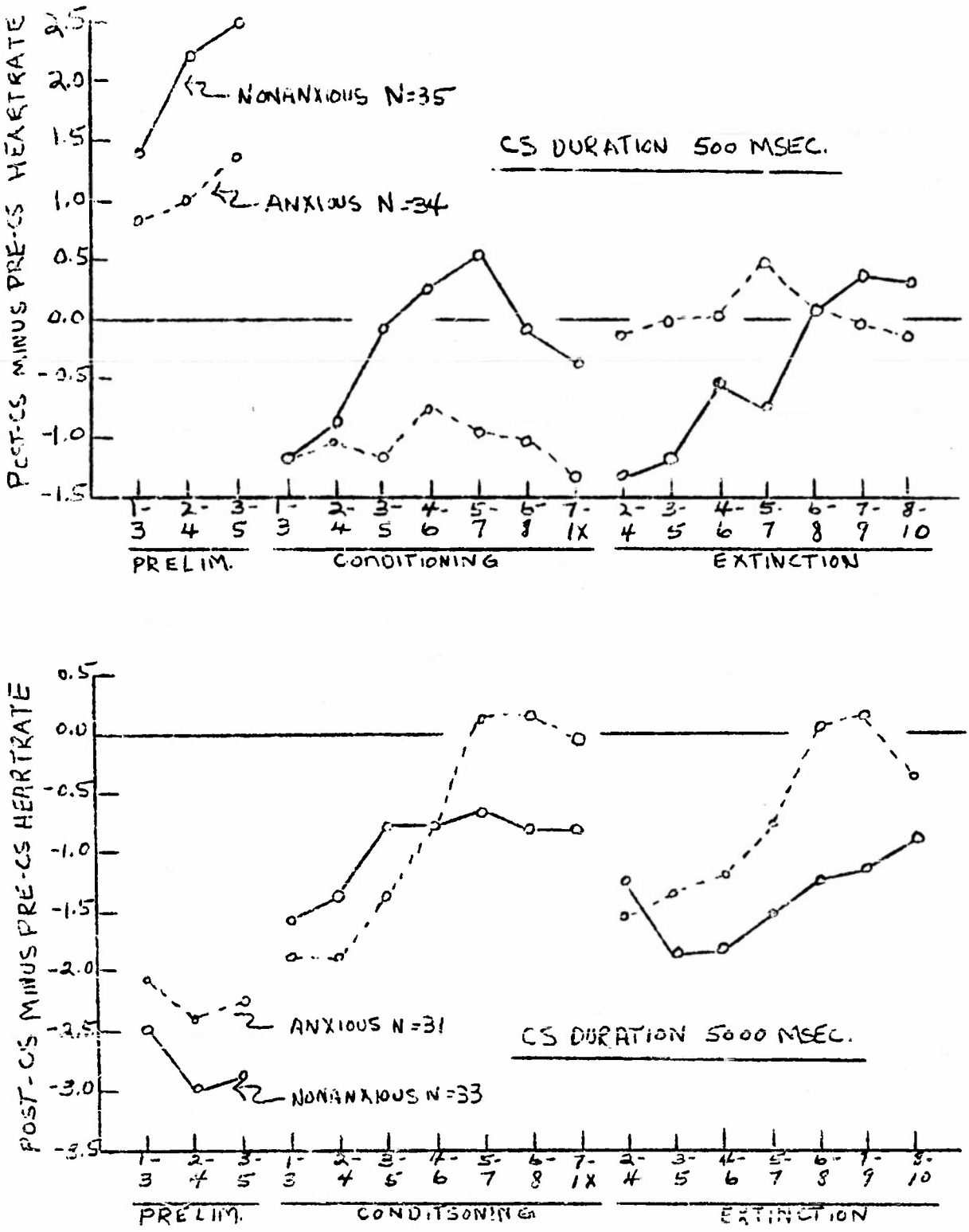

Fig. 1. Change in heart rate due to presentation of CS during preliminary trials (CS only), conditioning, and extinction.

Table 3

Effect of CS on Heart Rate (Post-CS minus Pre-CS rate)

on Trials 1 and 4 vs. Trials 5, 7, and 10 of Preliminary Series

Group	Trials	N	M	SD	t	p	Diff. 1&4 vs. 5,7,&10	t	p
500-HA	1 & 4	34	0.84	3.57	1.35	<.20	0.20	0.28	>.80
	5,7,&10	34	1.04	3.14	1.90	<.10			
500-LA	1 & 4	35	1.26	3.66	2.00	<.10	0.94	1.41	<.20
	5,7,&10	35	2.20	3.05	4.20	<.001			
5000-HA	1 & 4	31	-3.04	3.97	4.19	<.001	0.69	0.90	<.40
	5,7,&10	31	-2.35	3.44	3.74	<.001			
5000-LA	1 & 4	33	-3.02	4.02	4.25	<.001	-0.08	0.11	>.90
	5,7,&10	33	-3.10	5.47	5.47	<.001			

the scores on the first two trials vs. the last three trials for the four groups. These results indicate that the acceleration of heart rate by CS in the 500 msec. group was maintained throughout the preliminary trials; however, the changes in amount of acceleration as a function of practice were not significant. Similarly, the deceleration due to presentation of CS was maintained in the 5000 msec. groups, no significant changes in effect of CS during these trials being evidenced.

Response to CS during conditioning. Reference to Fig. 1 indicates that in the 500 msec. groups, there occurred a large drop in heart rate between the last preliminary trials and the early conditioning trials. ("Conditioning trials" refers to the test trials during conditioning. It will be recalled that an average of five actual conditioning trials, i. e., CS-UCS pairings, occurred between test trials, i.e., presentation of CS alone, during the conditioning series). In both the high and low anxious groups, the change involved a shift from acceleration to deceleration. A comparison of the means (of the means) for the last three trials of the preliminary practice and for the first three trials of conditioning shows that this drop was highly significant, $t = 3.76$, $p < .001$ for the 500-HA group, and $t = 7.11$, $p < .001$ for the 500-LA group.

Change in heart rate from the preliminary to the early conditioning trials was not so marked in the 5000 msec. groups. In both HA and LA subgroups, the rate was modified in the direction of decreased inhibition, but neither change was significant, $t = 0.46$, $p < .70$ for 5000-HA, and $t = 1.53$, $p < .20$ for 5000-LA.

The effect of CS-UCS interval was obviously highly significant with respect to these changes. The effect of anxiety level was insignificant.

As a result of these general effects, the response to CS in all groups during the early conditioning (test) trials was a retardation of heart rate. The effect of acquisition training, as seen in Fig. 1, was to decrease this inhibitory effect of the CS in all groups except 500-HA. The mean of the means in all groups for the first three and last three conditioning trials are presented in Table 4. (It should be noted that in both Fig. 1 and Table 4 the response on the first extinction trial was included in the computations of results at the termination of conditioning).

These data reveal that during the early portion of conditioning, the CS retarded heart rate significantly in all four groups. In two groups, 500-LA and 5000-HA, there was a significant recovery from this deceleration as a result of conditioning. In the 5000-LA group, the recovery was insignificant. And in the 500-HA group, the initial inhibitory effect increased, but not reliably. There appeared to be no consistent pattern of conditioned modification of heart rate relating to anxiety level. When the two 5000 msec. groups were combined, however ($M = 1.43$, $SD = 4.39$), their decrease in amount of inhibitory effect yielded a t of 2.59, $p < .02$, indicating that, at the 5000 msec. interval, conditioning had a consistent and reliable effect.

It is of interest to note that in three of the groups, the direction of change due to conditioning was the same as that reported by Zeaman and Wegner (15) and opposite to that reported by Notterman, Schoenfeld, and Bersh (5, 6). On the supposition that the direction of effect of conditioning depends upon the effect of the UCS at the time of its termination (cf. 15), and the further supposition that this direction is posi-

Table 4

Effect of CS on Heart Rate (Post-CS minus Pre-CS rate)

During Conditioning Trials

Group	Trials	N	M	SD	p	Diff. 1,2,3 vs. 7, 8, 1X	t	p
500-HA	1,2,3	34	-1.11	2.58	<.02	-0.59	0.88	<.40
	7,8,1X*	34	-1.70	3.14	<.01			
500-LA	1,2,3	35	-1.57	3.15	<.01	1.49	2.73	<.02
	7,8,1X	35	-0.08	0.16	<.90			
5000-HA	1,2,3	31	-1.98	3.46	<.01	1.58	2.37	<.05
	7,8,1X	31	-0.40	0.79	<.50			
5000-LA	1,2,3	33	-1.72	2.82	<.01	1.28	1.48	<.20
	7,8,1X	33	-0.44	0.87	<.40			

* First extinction trial

tive (accelerating) at the end of a very brief shock, the present results accord fairly well with expectation. It should be noted, however, that at the end of conditioning, the CS still appeared to have a slightly depressive effect in all groups, though it was inconsequential in three of the groups.

Response to CS during extinction. As the data in Fig. 1 indicated there was a tendency in all groups, when the extinction series was begun (extinction trials 2, 3, and 4), to shift in the direction opposite to that evidenced by the performance curves for the conditioning trials. Thus, the 500-LA group and the two 5000 msec. groups, all of which had shown decreasing retardation of heart rate during conditioning, showed increased inhibition during the early extinction trials. The 500-HA group, which showed an increasing retardation during conditioning, showed a substantial diminution of this inhibitory effect at the beginning of extinction. In other words, the initial effect of extinction was to modify the heart rate in the direction shown at the beginning of conditioning.

When the mean of means was computed for the last three conditioning (test) trials and the first three extinction trials, the decline of inhibition under the 500-HA condition yielded a t-value of 2.21, $p < .05$. Under the 500-LA condition, the increase of inhibition yielded a t of 2.49, $p < .02$. The t's for the increases of inhibition in the 5000-HA and 5000-LA groups were 1.59, $p < .20$, and 0.27, $p < .80$, respectively. When the three groups showing increased retardation were combined, $M = 1.08$, $SD = 4.46$, $t = 2.40$, $p < .02$. Thus, there is at least some basis for concluding that the shifts at the beginning of extinction were in the direction of

the kinds of response to CS characteristic of the early conditiong trials.

The picture presented in Fig. 1 with respect to the course of responses to CS during extinction bears a rather marked resemblance to that shown during conditioning. The three groups (500-LA, 5000-HA, and 5000-LA) whose deceleration decreased during conditioning tended to show a decline in deceleration during extinction also. The 500-HA group, whose response to CS during conditioning showed the least change (see Table 4) evidenced no consistent changes during extinction. The two groups, 500-LA and 5000-HA, that showed the most significant changes during conditioning appeared to show the greatest changes during extinction also.

A similar picture is presented by a comparison of the means of means for the early portion of the extinction series (trials 2, 3, and 4) with those for the terminal portion of the extinction series (trials 8, 9, and 10). These data are given in Table 5. They show that the only significant change occurring during extinction was that in the 500-LA group, which also showed the most significant change during conditioning.

At the end of the extinction series the effect of the CS was generally decelerative in all groups, but significantly so in only one, 5000-LA. The response to CS at the end of the extinction series was not reliably different from that at the end of the conditioning series, except in the 500-HA Ss, who showed a decrease in inhibitory effect that was significant at $p < .05$.

The explanation of the resemblance between the performance curves during the conditioning and extinction series can only be guessed at. Taken in conjunction with the changes in response between the last part of the conditioning series and the early part of the extinction series,

Table 5

Effect of CS on Heart Rate (Post-CS minus Pre-CS rate)

During Extinction Trials

Group	Trials	N	M	SD	t	p	Diff. 2,3,4 vs. 8,9,10	t	p
500-HA	2,3,4	34	-0.44	3.62	0.70	<.50	0.20	0.20	<.80
	8,9,10	34	-0.24	3.62	0.38	<.80			
500-LA	2,3,4	35	-1.86	3.45	3.14	<.01	1.86	3.22	<.01
	8,9,10	35	0.00	3.04	0.00	<.90			
5000-HA	2,3,4	31	-1.56	4.08	2.09	<.05	0.98	1.24	<.30
	8,9,10	31	-0.58	3.43	0.93	<.40			
5000-LA	2,3,4	33	-0.69	4.52	0.86	<.40	-0.35	0.37	<.80
	8,9,10	33	-1.04	2.61	2.26	<.05			

such results might be explained on the supposition that extinction had an immediate but temporary effect upon the heart rate itself, but not upon the fear generated by the CS. This persisting fear might then have produced a subsequent change in heart rate with successive "extinction" trials of the sort it produced during conditioning. An alternative explanation might be that the occurrence of a few successive nonreinforcements reduced fear early in the extinction series. But since a nonreinforcement had always been followed, in the conditioning series, by occurrence of shock on a succeeding trial, expectancy of shock (fear) may have increased again as extinction trials progressed, simply because S was kept in the experimental situation.

Comparison with previous findings. The present findings appear to indicate some sort of interaction between anxiety level and CS-UCS interval, in respect to heart-rate conditioning. No clear-cut main effects of these variables appeared, however, and the general trends were not in accord with any of those found in previous studies. Thus, Zeaman and Wegner (15) reported a conditioned acceleration of heart-rate when using a relatively short shock. In the present instance, the CS had a rather consistent decelerative effect. On the other hand, though Notterman et al. (5, 6) report a decelerative effect, they found that the degree of deceleration increased during conditioning. In the present study the amount of deceleration tended generally to decrease during conditioning.

It does not seem fruitful to speculate concerning possible causes of the differences between the results of the present study and those of previous studies, because of the many differences in procedure. For

instance, by contrast with the studies by Notterman et al., the present study involved delayed rather than trace conditioning, a much briefer UCS, preliminary trials involving UCS alone, a smaller number of preliminary trials using CS only, a ready signal, a light rather than tone as CS, and a considerably shorter inter-trial interval. Comparisons between the present study and that by Zeaman and Wegner are made difficult because of similar methodological differences, and also because of differences in computational procedures.

Certain aspects of the present results suggest that the human heart rate is conditionable. The direction and degree of the conditioning effects, and the specific factors influencing them, are far from clear, however. In general, the present study cannot be considered as strongly confirming the generality of the findings of previous investigators of human heart rate conditioning.

Summary

An attempt was made to condition the heart rate of 65 high anxiety Ss and 68 low anxiety Ss, using a delayed conditioning procedure, with shock as the UCS and a light as the CS. For approximately half of the Ss in each group, the duration of the CS was 500 msec.; for the other half, the duration of the CS was 5000 msec.

Presentation of the UCS alone during preliminary trials appeared to accelerate heart rate in all groups. Presentation of the CS alone during preliminary trials resulted in a slight and insignificant acceleration of heart rate in the 500 msec. groups, but a significant deceleration in the 5000 msec. groups. During early conditioning trials, the CS produced

a significant deceleration of heart rate in all groups. After 40 conditioning trials (32 actual conditioning trials and eight test trials), the magnitude of this inhibitory effect had decreased in three of the groups, the exception being the 500 msec. high anxiety group. Early in extinction, all groups showed a modification of heart rate in the direction characteristic of response to CS at the beginning of conditioning. Curves of performance during ten extinction trials tended to resemble those obtained for the conditioning series.

Although the results gave some suggestion that conditioning occurred, they were not related in any clear way to either level of anxiety or CS-UCS interval. Nor were the results in general similar to those reported by previous investigators. Consequently, the present study does not strongly confirm the generality of previous findings with respect to the conditioning of the human heart rate.

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